OUR EXPERIENCE WITH ST.JUDE PROSTHETIC MITRAL VALVE REPLACEMENT IN 179 CONSECUTIVE PATIENTS

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CERTIFICATE

This is to certify that the dissertation entitled "OUR EXPERIENCE WITH St.JUDE PROSTHETIC MITRAL VALVE REPLACEMENT IN 179 CONSECUTIVE PATIENTS" presented here is the original work done by Dr.M.Gopal in the department of cardiothoracic surgery, Govt.General hospital,Madras Medical college, Chennai 600003,in partial fulfillment of the university rules and regulations for the award of Mch cardiothoracic degree under our guidance and supervision during the academic period from 2004-2007.

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INTRODUCTION

Rheumatic heart disease plays a major debilitating role in Indian population causing high morbidity and mortality, affecting the population in their productive phase of life. It impairs the quality of life and leads to psycho-social and economic burden to their family and the society. With the clinical introduction of cardiac valvular prosthesis in 1960, valve replacement has saved a number of lives affected by hemodynamically significant valvular disease. However, prosthetic valve replacement is not with out danger and it became evident that "valve replacement is simply exchanging one disease for another"(1). In spite of tremendous strides toward perfecting materials and design, the ideal valve remains elusive. Thromboembolism continues to be a major cause of morbidity and mortality in patients who have undergone mechanical valve replacement. Recent surgical trend in treating Rheumatic mitral valve disease is to Repair the valve. If Repair is not feasible, then valve replacement is still undertaken knowing the complications unique to prosthetic valve replacement in the interest of saving the life.

ANATOMY OF MITRAL VALVE

The Mitral valve apparatus consists of leaflets, annulus, chordae tendineae, papillary muscles, and left ventricle. Mitral valve and its apparatus plays an important role in maintaining the left ventricular geometry and function.

Annulus:-

The Mitral annulus is the area of leaflet attachment to muscular fibers of the atrium and ventricle. The annulus is pliable, permitting sphinteric contraction and decreases in diameter during each systolic contraction by approximately 26%. The orifice of the Mitral valve also changes shape, from elliptical during ventricular systole to circular during late diastole. This flexibility increases leaflet coaptation during systole and maximizes orifice area during diastole. Changes in size and shape of the annulus result from relaxation and contraction of the basoconstrictor muscles (bulbospiral and sinospiral bundles). In the horizontal plane the annulus is saddle-shaped. Anteriorly, the annulus is attached to the fibrous skeleton of the heart, extending between the two fibrous trigones. This limits its pliability and its capacity to dilate with mitral regurgitation (MR). The posterior annulus is more flexible and is not attached to rigid surrounding structures. This is confirmed by the clinical observation that dilation of the annulus occurs posteriorly with MR.

Anatomical knowledge of the mitral annulus and surrounding structures is critical to avoid inadvertent damage during mitral surgery. The circumflex coronary artery runs laterally around the mitral annulus in the posterior atrioventricular groove. The coronary sinus runs more medially in the same groove. The artery to the atrioventricular node, usually a branch of the right coronary artery, runs parallel and close to the annulus of the anterior leaflet near the posteromedial commissure. The aortic valve is situated between the anterior and posterior fibrous trigones. The bundle of His is located near the posterior trigone.

Leaflets:-

The mitral valve has two leaflets, the anterior (aortic) and posterior (mural) leaflets. The leaflets are attached to the mitral annulus and at its free border to the papillary muscles by primary and secondary chordae. The anterior mitral leaflet is in direct continuity with the fibrous skeleton of the heart. This leaflet is contiguous with the left and noncoronary cusps of the aortic valve and the area below the intervening aortic commissure, termed the fibrous subaortic curtain. Although the anterior leaflet occupies only 35% to 45% of the annular circumference, its leaflet area is almost identical to that of the posterior leaflet.

The posterior leaflet is rectangular. The free margin of the posterior leaflet has two clefts that divide the posterior leaflet into three scallops: the largest or middle scallop, the posteromedial scallop, and the anterolateral scallop. Fan-shaped chordae

insert into and define the clefts between the individual posterior scallops. Motion of the posterior leaflet is more restricted than that of the anterior leaflet; however, both mitral leaflets contribute importantly to effective valve closure. The valvular surface area is bigger compared to the orifice area. The ratio is close to 2:1. This difference exaggerates even more during systole because of the sphincter action of annulus which reduces further the orifice area.

There are three phases in valve orifice closure.

1) Edge to edge meeting between leaflet.

2) Upward bulge of leaflet.

3) Final phase maximal cooptation of leaflet almost they lie vertically.

The surface of the mitral leaflet is divided into three zones corresponding to areas of chordal insertion and leaflet coaptation. The rough zone is the leading edge of the anterior and posterior mitral leaflets. This zone is the contact surface of the mitral leaflets during systole. The clear zone is peripheral to the rough zone and represents most of the body of the leaflet; this portion of the mitral valve billows into the atrium during ventricular contraction. The basal zone, between the clear zone and the annulus, receives the insertion of the basal chordae tendineae (tertiary chordae), which originate directly from the trabeculae of the left ventricle. The basal zone is found only on the posterior leaflet.

Chordae Tendineae :-

The chordae tendineae are chords of fibrous tissue that attach the mitral leaflets to either the papillary muscles or the left ventricular free wall. They often subdivide and interconnect before they attach to the leaflets. The chordae are divided into primary , secondary , and tertiary chordae. Primary chordae attach directly to the fibrous band running along the free edge of the leaflets. These chordae ensure that the contact surfaces (rough zone) of the leaflets coapt without leaflet prolapse or flail. Secondary chordae attach to the ventricular surface of the leaflets at the junction between the rough and clear zones. These chordae contribute to ventricular function. Secondary chordae helps the ventricle to contract in an efficient cone-shaped fashion; when secondary chordae are excised, the left ventricle assumes a globular shape. Tertiary chordae are unique to the posterior leaflet. They arise as strands directly from the left ventricular wall or from small trabeculae to insert into the ventricular surface of the leaflet near the annulus.

Papillary Muscles :-

The anterolateral and posteromedial papillary muscles each supply chordae tendineae to both leaflets. The two groups of papillary muscles support the anterolateral and posteromedial commissures and arise from the junction of the apical and middle thirds of the ventricular wall. The anterolateral papillary muscle receives a dual blood supply from the anterior descending coronary artery and either a diagonal branch or a

marginal branch of the left circumflex artery. The posteromedial papillary muscle receives its blood supply from either the left circumflex artery or a distal branch of the right coronary artery. Because of the single blood supply to the posteromedial papillary muscle, infarction of the posteromedial papillary muscle is much more common. Blood supply comes through a large central artery at its base from epicardial vessel then dividing in to network to supply the papillary muscle. The occlusion of large central artery severely damages the entire papillary muscle, due to lack of collateral supply and leading to necrosis of the papillary muscle which leads to mitral regurgitation.

Left Ventricle :-

The posterior left ventricular wall and papillary muscles play an important role in leaflet coaptation and valve competence. Papillary muscles are aligned parallel to the ventricular wall and attach via chordae to the free edges of the valve leaflets. These muscles project from the trabeculae and may be single, bifid, or a row of muscles arising from the ventricular wall. During isovolumetric contraction the mitral leaflets are pulled downward and together by this interaction. Ventricular dilatation may affect the alignment and tension on the papillary muscles and valve competence.

Atrial Fibrillation :-

This is the Commonest rhythm disturbance that accompanies mitral valve disease. In Atrial fibrillation walls of the atria shudder fast and atrial contraction will not be present. At the same time ventricular rate is fast and irregular due to chaotic

bombardment of AV node by more than 700 impulse per minute from the atria. The trigger of an AF is a focus of acute localized wall stretch, which when on chronic remodeling occurs with histological changes. Occurrence of AF has been consistently related to the size of the left atrium. Univariate analysis in one study revealed that the incidence of AF was 3% when the left atrial diameter was less than 4.0 cm but increased to 54% if the left atrial diameter was more than 4.0 cm, thus explaining the highest incidence in patients with mitral valve disease, which causes maximum left atrial enlargement.(2)

For AF patients, SA node histology indicates severe degeneration of normal pacemaker tissue(3)

It is postulated that fibrosis and degeneration of the atrial myocardium in valvular heart disease, especially of rheumatic etiology disturbs impulse propagation in the atria and leads to AF(4)

Atrial fibrosis probably contributes to persistent AF after valvuloplasty or valve replacement/repair. In patients with valvular disease, AF also occurs more frequently with mitral valve calcification, mitral valve prolapse and following valve replacement surgery.(5,6)

Atrial wall shows myofibrillar changes with increased fibrous tissue deposition, leading to haphazard electrical activity. Mechanically there is a loss of atrial

contribution to stroke volume by up to 30%. Combination of loss of atrial kick and fast ventricular rate (loss of diastolic filling time) occur plus loss of normal presystolic closure timing of atrioventricular valves due to fast ventricular rate and absent atrial contraction, all of which decreases cardiac output and mitral regurgitation may occur.

Dilatation of atrial wall leads to increased secretion of Atrial natriuretic peptide. Fast heart rate precipitates congestive cardiac failure by tachycardia mediated cardiomyopathy (7) there by AF per se is responsible for overt heart failure by increasing myocardial metabolism by provoking rapid myocardial contraction leading to exhaustion. Dilated atrium, loss of atrial contraction are factors for clot formation. After the onset of AF, stroke is the most feared and calamitous complication. Compared to sinus rhythm AF carries an increased mortality of about 1.5- 1.9 independent of heart disease and age and part of this increased mortality is likely due to stroke in its various presentation.(8,9)

Evidence based Treatment

The Committee on Management of Patients with Valvular Disease was given the task of reviewing and compiling this information base and making recommendations for diagnostic testing, treatment, and physical activity. These guidelines follow the format established in previous American College of Cardiology/American Heart Association (ACC/AHA) guidelines for classifying indications for diagnostic and therapeutic procedures :

Class I : Conditions for which there is evidence and / or general agreement that a given procedure or treatment is useful and effective.

Class II : Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment IIa. Weight of evidence/opinion is in favour or usefulness/efficacy

IIb. Usefulness/efficacy is less well established by evidence/opinion.

Class III : Conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful and in some cases may be harmful.

Indication for Mitral Valve Replacement

Indications for valve replacement pertain to those types of valve that are unlikely to be repaired or which have been shown to have poor long-term success after previous intervention.

Mitral Stenosis

Mitral stenosis is almost exclusively caused by rheumatic fever. The pathologic changes in rheumatic valvulitis are mainly fusion of the valve leaflets at the

commissures; shortening and fusion of the chordae tendinae; and thickening of the leaflets due to fibrosis, contraction, and calcification.

Stenosis usually develops one or two decades after the acute illness of rheumatic fever with no or slow onset of symptoms until the stenosis becomes more severe. Limitation of exercise tolerance is usually the first symptom followed by dyspnea that can progress to pulmonary edema. New onset atrial fibrillation and risk for thromboembolism, hemoptysis, and pulmonary hypertension are other common symptoms in patients with mitral stenosis.

The diagnostic workup of the symptomatic patient with mitral stenosis should include a complete cardiac catheterization, including coronary angiography in any patient over the age of 40. Under the age of 40, echocardiographic findings of the mitral valve is enough in most symptomatic patients for the definition of mitral valve pathology unless there is a history of chest pain or coronary artery disease.

In general, operation is prescribed when the mean valve area is 1.0 cm^2 or less (normal mitral valve area: 4–6 cm^2 - however, with a "mixed" lesion of mitral stenosis and mitral regurgitation, the valve area in symptomatic patients occasionally may be as large as 1.5 cm^2. Asymptomatic patients are generally not considered for surgery but it is

in the asymptomatic patients with significant hemodynamic mitral stensosis , still surgery is considered.

ACC / AHA RECOMMENDATIONS FOR MITRAL VALVE REPLACEMENT IN MITRAL STENOSIS

1. Patients with moderate or severe MS (mitral valve area ≤1.5cm^2) and NYHA functional Class III-IV symptoms who are not considered candidates for percutaneous balloon valvotomy or mitral valve repair – Class I

2. Patients with severe MS (mitral valve area ≤ 1 cm^2) and severe pulmonary hypertension (pulmonary artery systolic pressure > 60 to 80mm Hg) with NYHA functional Class I-II symptoms who are not considered candidates for percutaneous balloon valvotomy or mitral valve repair – Class IIa.

Mitral Regurgitation

The etiology of mitral regurgitation is very diverse. Except in cases of acute ischemic mitral regurgitation and endocarditis, where indications are more straightforward, indication generally for regurgitative lesion are complex. Etiology of

regurgitation are degenerative (mitral prolapse, ruptured/elongated chordae), rheumatic, infectious, and ischemic diseases of the mitral valve. Most of these are now amenable to mitral valve repair and reconstruction .

For any of the preceding major pathologic subsets, indications for surgery in patients with mitral regurgitation vary from the asymptomatic patient with an enlarging but well functioning left ventricle and atrium to severely depressed left ventricular function. Any symptomatic patient with significant mitral regurgitation (3+ to 4+) should be operated on, and operation should be considered in any relatively symptom-free individual if there is objective evidence of left ventricular deterioration and significant increase in left ventricular end-systolic and end-diastolic volumes.

Regurgitation through the valve is usually measured with Doppler echocardiography, but MRI is another noninvasive technology for measuring the regurgitant flow and can provide measurements of ventricular end-diastolic/systolic volumes and ventricular mass. Left ventricular angiography with coronary angiogram can be helpful but is otherwise indicated for evaluating the coronary arteries preoperatively in patients older than 40 years.

It is important to stress that ejection fraction is a poor indicator of left ventricular function in patients with mitral regurgitation. Depressed cardiac output (< 40%)

therefore usually indicates severe left ventricular dysfunction, and results of surgery are not as favorable in these patients as they are in patients with normal ventricles. Compared to ejection fraction, measurements of end-systolic volume and diameter are more reliable noninvasive parameters to evaluate the status of the left ventricle and determine the optimal time for operation .

ACC / AHA **RECOMMENDATIONS FOR MITRAL VALVE SURGERY IN NONISCHEMIC SEVERE MITRAL REGURGITATION.**

1. Acute symptomatic MR in which repair is likely – Class I

2. Patients with NYHA functional Class I, III, or IV symptoms with normal LV function defined as ejection fraction > 0.60 and end-systolic dimension < 45 mm – Class I.

3. Symptomatic or asymptomatic patients with mild LV dyfunction, ejection fraction 0.50 to 0.60, and end-systolic dimension 45 to 50 mm – Class I.

4. Symptomatic or asymptomatic patients with moderate LV dysfunction, ejection fraction 0.30 to 0.50, and/or end-systolic dimension 50 to 55 mm – Class 1.

5. Asymptomatic patients with preserved LV function and atrial fibrillation – Class IIa.

6. Asymptomatic patients with preserved LV function and pulmonary hypertension (pulmonary artery systolic pressure > 50mm Hg at rest or > 60 mm Hg with exercise) – Class IIa

7. Asymptomatic patients with ejection fraction 0.50 to 0.60 and end-systolic dimension < 45 mm and asymptomatic patients with ejection fraction > 0.60 and end-systolic dimension 45 to 55 mm – Class IIa.

8. Patients with severe LV dysfunction (ejection fraction < 0.30 and/or end-systolic dimension > 55 mm) in whom chordal preservation is highly likely – Class IIa.

9. Asymptomatic patients with chronic MR with preserved LV function in whom mitral valve repair is highly like – Class IIb.

10. Patients with MVP and preserved LV function who have recurrent ventricular arrhythmias despite medical therapy – Class IIb.

11. Asymptomatic patients with preserved LV function in whom significant doubt about the feasibility of repair exists – Class III.

REVIEW OF LITERATURE

History of valve surgery :-

Cardiac valve repair or replacement under direct vision awaited the development of the heart-lung machine. With the contribution by John Gibbon to the the development of the heart-lung machine, valve replacement become feasible. The first successful prosthetic mitral valve replacement was done by Nina Braunwald in 1960. (10)This was followed by Starr and Edwards, replacing the mitral valve with a caged ball valve in September 1960, from then problems of post operative low cardiac output, thromboembolism, anticoagulation related bleeding came to the fore.(11)

Due to its improved flow dynamics and decreased thromboembolic complications the first clinical trial with the St.Jude medical prosthesis was initiated in April 1979.This is a low profile bileaflet tilting disc Valve constructed entirely (except for the sewing ring made of polyester) with pyrolytic carbon. The graphite substrate of leaflet is tungsten impregnated for radiopacity.(12)

Central flow design offers a lower transvalvular gradient , durable ,thromboresistance, low incidence of valve related adverse events. Of the more than 70 mechanical valves in clinical usage the St.Jude medical valve has been the most successful and has been the gold standard for mitral valve replacement(13,14,15,16,17) in all age groups, from infant to the elderly . (18,19,20,21,22,23)

According to Crawford etal the immediate operative mortality for valve replacement may not be related to the type of prosthesis used (12).

Lim etal reported from their study that Carbomedics and St Jude valve had no significant difference in early & midterm clinical outcomes. The choice with respect to valve type can be based on other than patient outcomes.(24).

Eric etal also confirmed, no significant differences exists with regard to thromboembolic and hemorrhagic complications between Carbomedics & St jude prosthetic valves.(25)

Camilleri etal in their study concluded, no significant difference exists between St. Jude Medical and Sorin Bicarbon valves over a 4-yr period of follow-up with respect to morbidity, mortality and event free survival rates (26).

Murday etal compared Starr Edwards and St. Jude prostheses in the aortic and mitral positions. They found no differences in complication rate or symptomatic improvement between the Starr Edwards and St. Jude valve prostheses in either aortic or mitral position. Left ventricular function had such a impact on long-term survival that it overwhelmed any differences between different prosthetic valve designs.(27).

Ye-Ying Cen etal in their analysis of Carpentier-Edwards bio prosthesis and St Jude mechanical valve found , 10-year survival was not statistically different between the two but factors predicting adverse survival after mitral valve replacement

were older age, lower ejection fraction, presence of class IV congestive heart failure, coronary artery disease, renal disease, smoking history, hypertension, combined valve surgery, and redo heart surgery. Choice of biologic or mechanical prosthesis does not significantly affect long-term patient survival after mitral valve replacement.(28).

Douglas etal from their study with St. Jude or porcine mitral valve replacement found no statistical differences in symptomatic improvement, mortality ,complications and actuarial survival.(29)

According to Ikonomidis etal valve replacement patients do not survive in parallel to normal population. Factors influencing the operative mortality are age, previous valve replacement, cardiac function, coronary artery disease these factors influence on operative mortality than the type of valve prosthesis implanted. Late death is unrelated to prosthetic valve, and the factors influencing the long term outcome is higher NYHA class, concominant CABG, increasing age. (14)

De Santo etal in their study with young rheumatic women identified atrial fibrillation as a risk factor for late mortality, whereas higher left ventricular ejection fraction at 12 months postoperative proved to be a protective factor.(30).

Remadi etal in their study identified age and sex as significant risk factors for valve-related mortality and that higher age, female sex, higher NYHA class, and atrial fibrillation had significant correlation with overall higher mortality(15)

Fernandez etal from their study reported, that risk for inhospital mortality included older age, female gender , higher preoperative left ventricular end-diastolic pressure, previous cardiac operation , longer aortic crossclamp time , and longer cardiopulmonary bypass time . Risk factors for late death included older age, lower preoperative ejection fraction , longer aortic crossclamp time, longer cardiopulmonary bypass time , previous cardiac operation, and higher preoperative functional class .(16)

According to Arom etal thrombo embolic events may occur some times because of valve thrombosis, but in general they arose from adjacent part of sewing ring. The embolic incident can occur without evidence of associated valve dysfunction. (31)

Duncan etal noted the factors that led to thromboembolism were enlarged cardiac chamber(S), calcification in chamber walls or annulus, dysrhythmias and techniques of operative implantation.(32)

Debétaz etal prognosticated the Independent preoperative risk factors for embolism, were cardiac failure as an indication for operation and history of prior systemic embolism (33)

Emery etal observed that thromboembolism is more common in the immediate postoperative period and has a continuous hazard phase through out the life of the patient. Thromboembolism is very high in early postoperative period in the face of newly implanted sewing ring which is a nidus for emboli production.(13)

De Santo etal observed that Atrial fibrillation was significantly associated with a higher incidence of thromboembolic events.(30)

Remadi etal found valve replacement patients had persistent problem with anticoagulation management even after 10 years follow up which alter the quality of life. Hence the recommendation for mitral plasty techniques, whenever it is technically feasible. Light anticoagulant treatment is now recommended for patients in sinus rhythm to decrease the serious hemorrhagic complications.(15).

Emery etal in their study observed that anticoagulation related hemorrhage is more common in the first postoperative year. Patient related factors are important than valve related factors. Hence they advised the target INR for patients to be individualised .(13)

Hering etal in their study found out, thromboembolism following AVR was significantly lower than after MVR. Comparing three different intensities of oral anticoagulation in valve replacement patients , they found out that the incidence of thrombo embolism and bleeding complications was comparably low in all INR strata and recommended to reevaluate the intensity of anticoagulation in patients with the SJM valve.(34)

Lafci etal in their study with bileaflet valve thrombosis, found out majority of patients had subtherapeutic anticoagulation level leading to valve thrombosis and they presented in NYHA class IV symptoms clinically. (35)

Shapira etal from their study inferred that bileaflet valves have delicate mechanism and may lead more easily to leaflet immobilization, even with a relatively small clot. (36)

Deviri etal from their study observed, majority of the thrombus was at the hinge site, causing impairment of both leaflet motion. Amount of thrombotic material needed to cause interruption of leaflet motion in bileaflet valves is minimal, especially if it catches the hinges of the valve. A small thrombus may even entrap the hinges of both leaflets(37)

Laplace etal noted higher incidence (about 10%) prosthetic valve thrombosis in early postoperative period with in nine days. Pre operative atrial fibrillation immediate postoperative impaired left ventricle ejection fraction (<50%), and high dose protamine plus inadequate postoperative anticoagulation are implicated. Formation of thrombus is explained by Virchows triad -cardiac surgery, mechanical valve, extracorporeal circulation damages the native tissues, while artificial surfaces alters the rheology with stagnation of blood , reducing the flow velocity. A hypercoagulable state exists due to platelet leucocyte , and coagulation factor activation, fibrinogen

maintaining the vicious cycle. Hence in their study they advocated the need for transesophageal echocardiogram in the immediate post operative period.(38).

Cowgill etal observed in their study that predominant cause of early prosthetic valve endocarditis (<60 days) is caused by S.Epidermiditis, followed by S.Aureus due to perioperative contamination. Late prosthetic valve endocarditis(>60 days) is predominated by Streptococcal infection.(39)

Arom etal observed that the incidence of prosthetic valve endocarditis can be reduced by prophylactic antibiotics prior to valve replacement, shorter operating time, and improved antibiotic prophylaxis for minor or invasive procedures.(31)

Wells etal observed patient in AF had a lower survival rate compared to sinus rhythm patients. The onset of AF is an indicator of severity and chronicity of disease due to its association with poor LV function. Early surgery after the onset of AF has its benefits . Lower survival was seen in patients who persist in AF in post operative period. Left atrial size were significantly larger in patients who were in atrial fibrillation even after surgery.(40)

Mehta etal reported that in patients in sinus rhythm, the prevalence of left atrial clot was 0% in mitral regurgitation and 14.3% in patients with mitral stenosis.(41)

Diker etal in their study reported, AF has been found in 40% of cases with mitral stenosis and 25% of cases with mitral regurgitation.(42)

Falk etal in their study reported Atrial fibrillation is responsible for more than 85% of systemic thromboembolism from the heart.(43)

Kannel etal from their study confirmed that when associated with mitral valve disease, AF has the highest stroke risk about seventeen times greater than in unaffected controls. In comparison, in patients with nonvalvular heart disease, the risk is increased five-fold.(44)

Jorgensen etal reported Stroke in patients with AF is associated with 70% increased mortality and very poor neurological and functional outcome. The recurrence rate in the first year after the initial stroke varies between 13% and 32%.(45)

Morris reported that Lifetime stroke recurrence rates, especially for patients with AF associated with mitral valve disease, may be as high as 30%–75%.(46)

Shuhaibera etal noted in their meta- analysis of 29 published studies, a strong evidence at 30 day and overall survival favouring mitral valve repair in rheumatic mitral valve disease. The risk of thrombo embolism is also lower in Repair group.(47)

Carpentier etal had shown good long term results with repair of young Rheumatic patients with mitral insufficiency. The out come is superior to valve replacement procedures. Repair had a low hospital mortality rate and an acceptable rate of reoperation with minimal risk of thromboembolic events in repair compared to replacement.(48)

According to Chowdhury etal total chordal preservation is possible in the large majority of rheumatic patients and this helps to preserve left ventricular function .(49)

Miller etal described that preservation of all or part of the posterior leaflet and its chordae does not interfere with prosthetic valve function and it helps to reduces the risk of ventricular rupture and enhances the survival after mitral valve replacement. (50)

Jun Zhang etal reported that mortality following LV rupture in mitral valve replacement was 61.5% and advised careful removal of diseased valve , decalcification, and prosthesis selection should minimize the incidence of left ventricular rupture. Immediate diagnosis and urgent surgical intervention are crucial for successful out come.(51)

Hillman etal reported that young adults (< 21 years) with rheumatic heart disease had good result with Mitral repair surgery in that it allows annular growth with good long term functional result. (52)

Yau etal described that Mechanical valves minimize reoperation rate but at the cost of limiting the survival and increases the thromboembolic complications. Patients undergoing valve repair had improved late survival independent of their preoperative characteristics. Rheumatic mitral valves should be repaired when

technically feasible, accepting a small risk of reoperation, to maximize survival and reduce morbidity (53)

AIMS AND OBJECTIVES

1) To study the complications associated with Mechanical Mitral valve replacement.

2) To Study the influence of AF on outcome following Mitral valve replacement.

MATERIALS AND METHODS

This prospective study was conducted between June 2005 to December 2006 on 179 consecutive patients undergoing valve replacement with St. Jude prosthetic Mitral valve. Valve lesion other than tricuspid valve was not included in this study. Similarly ischaemic mitral regurgitation was not included. Previous open heart surgery patients were not included in this study. St.Jude bileaflet low profile prosthetic valve Masters series MJ 501 with standard polyester cuff was used in all patients. Male patients 40 years and above and female patients 45 years and above without risk factor for ischemic heart disease and those patients who are at high risk for ischemic heart disease were subjected to coronary angiogram pre operatively.

Operative technique:-

Surgery was conducted with a midline sternotomy incision, standard cardiopulmonary bypass with Aortic , bicaval cannulation, core was cooled to 28*C. Heart was arrested with hyperkalemic blood cardioplegia, with topical ice slush being used to cool myocardial temperature .Left atrium was opened parallel to the interatrial groove in those patients who have isolated mitral pathology .In ASD, Tricuspid regurgitation, LA myxoma patients Right atrium was also opened . Surgery was conducted after inspecting the mitral valve for suitability for chordal preservation . St.Jude prosthetic mitral valve MJ501 masters series was used in all patients and suturing was done with 2 0 Ethibond sutures with plegets using a horizontal interrupted

mattress technique. Continuous suture technique for those annulus not severly diseased or calcified was employed too .The valve was placed in intra annular position.

Patients were ventilated electively and ionotropes used depending up on the hemodynamic needs. On first post operative day heparin was given along with oral anticoagulant (Acenocoumarol) and continued until the target INR of 2.5 to 3.0 reached, then heparin stopped. Anticoagulant dose was regulated by surgeons during hospitalization and during subsequent follow up in the outpatient department. Patients came for regular follow up every 15 days for drug collection and for clinical assessment. In those patients who were symptomatic or clinical suspicion of deterioration were present subjected to detailed evaluation. 75% of the patients were still in follow up .

TABLES

Table : 1

Age group (in yrs)

	Frequency	Percent
<10 yrs	1	.6
11-20 yrs	41	22.9
21-30 yrs	64	35.8
31-40 yrs	45	25.1
41-50 yrs	23	12.8
51-60 yrs	5	2.8
Total	179	100.0

Table : 2

Sex

	Frequency	Percent
Female	109	60.9
Male	70	39.1
Total	179	100.0

Table : 3

Etiology

	Frequency	Percent
Congenital	1	0.6
Degenarative	5	2.8
Rheumatic	173	96.6
Total	179	100

Table : 4

Diagnosis

	Frequency	Percent
MR	46	25.7
MRS	20	11.2
MS	84	46.9
MS/MR	29	16.2
Total	179	100.0

Table : 5

Co morbidity-details

	Frequency	Percent
ACUTE MR	2	5.4
ASD	5	13.5
CAD	3	8.1
INF. ENDOCARDITIS	2	5.4
LA CLOT	21	56.8
LAMYXOMA	1	2.7
TR -SEV	3	8.1
Total	37	100.0

Table : 6

Previous Sx

	Frequency	Percent
CMC	22	12.3
NIL	157	87.7
Total	179	100.0

Table : 7

Elective/Emergency

	Frequency	Percent
ELE	176	98.3
EMER	3	1.7
Total	179	100.0

Table : 8

NYHA-Preop

	Frequency	Percent
C2	4	2.2
C3	142	79.3
C4	31	17.3
C5	2	1.1
Total	179	100.0

Table : 9

AF/NSR-Preop

	Frequency	Percent
AF	70	39.1
NSR	109	60.9
Total	179	100.0

Table : 10

EF%-Pre op

	Frequency	Percent
50 and above	170	95.0
30-49	9	5.0
Total	179	100.0

Table : 11

Procedures

Procedures	Frequency	Percent
CMC + MVR	2	1.1
ISOLATED MVR	144	80.4
MVR+ASD	5	2.8
MVR+CABG	3	1.7
MVR+DEVEGA	3	1.7
MVR+LAMyxoma excision	1	0.6
MVR+LA.CLOT removal	21	11.7
Total	179	100.0

Table : 12

Classical / Chordal Preservation

Procedure	Frequency	Percent
Chordal	83	46.4

Classsical	96	53.6
Total	179	100.0

Table : 13

valve size

Size mm	Frequency	Percent
25	66	36.9
27	92	51.4
29	21	11.7
Total	179	100.0

Table : 14

Post op NSR / AF:

	Frequency	Percent
AF	67	37.4
NSR	110	61.5

Pacing	2	1.1
Total	179	100.0

Table : 15

EF%-Post op

	Frequency	Percent
50 and above	129	77.7
30-49	37	22.3
Total	166	100.0

Table : 16

EF%-Pre op * EF%-Post op Crosstabulation (AMONG CLASSICAL REPLACEMENT GROUP)

		EF%-Post op		Total
		50 and above	35-49	
EF%-Pre op	50 and above	49 61.3%	31 38.8%	80
	35-49		3 100.0%	3
Total		49	34	83

Table : 17

EF%-Pre op * EF%-Post op Crosstabulation (AMONG CHORDAL PRESERVATION GROUP)

		EF%-Post op		
		50 and above	35-49	Total
EF%-Pre op	50 and above	78 96.3%	3 3.7%	81
	35-49	2 100.0%		2
Total		80	3	83

Table : 18

NYHA-Post op

	Frequency	Percent
	14	7.8
C1	19	10.6
C2	117	65.4
C3	28	15.6
C4	1	6
Total	179	100.0

Table : 19

Complications

	Frequency	Percent
No complication	141	78.8
Valve thrombosis	6	3.4
Thrombo- Embolism	2	1.1
Prosthetic valve endocarditis	2	1.1
Bleeding event	4	2.2
Myocardial failure-LCO	8	4.5
DecubitusUlcer	2	1.1
Haematuria	1	6
LA Appendage tear	1	6
LV rupture	2	1.1
Mediastinitis	1	6
Superficial wound infection	4	2.2
Ventricular arrythmia	1	6
Bleeding reexploration	2	1.1
CCF	1	6
intraopCoagulation failure	1	6
Total	179	100.0

Table : 20

Complications (AMONG 'NSR' PATIENTS)

	Frequency	Percent
No complication	94	86.2
Embolism	2	1.8
Prosthetic valve endocarditis	2	1.8
Bleeding event	3	2.8
Myocardial failure-LCO	1	.9
Ulcer	1	.9
Haematuria	1	.9
Mediastinitis	1	.9
Superficial wound infection	3	2.8
Ventricular arrythmia	1	.9
Total	109	100.0

Table : 21

Complications (AMONG 'AF' PATIENTS)

	Frequency	Percent
No complication	47	67.1
Valve thrombosis	6	8.6
Bleeding event	1	1.4
Myocardial failure-LCO	7	10.0
Ulcer	1	1.4
LA Appendage tear	1	1.4
LV rupture	2	2.9
Superficial wound infection	1	1.4
Bleeding reexploration	2	2.9
CCF`	1	1.4
Coagulation failure	1	1.4
Total	70	100.0

Table : 22

Cause of death

Cause of death	Frequency	Percent
Valve thrombosis	3	1.7
Myocardial failure	8	4.5
Bleeding- coagulation failure	1	0.6
Prosthetic valve endocarditis	2	1.1
LV rupture	2	1.1
LA appendage tear	1	0.6
Ventricular arrythymia	1	0.6
Mediastinits-strenal infection	1	0.6
Total	19	10.8

OBSERVATION

Of these 179 patient, operated with St.Jude prosthetic mechanical mitral valve replacement, between June 2005 to December 2006 isolated MVR was done in 144(80.4%).When LA clot removal was combined with MVR procedure it accounts to 165(92.1%). Depending up on the severity of subvalvuar lesion, classical or chordal preservation was done. The chordal preservation could be total (Anterior and posterior leaflet) or partial (Posterior alone).

Aortic cross clamp time ranged from minimum of 38 min to maximum of 110 min and Cardio pulmonary bypass time ranged from minimum of 52min to maximum of 140 min.

Table 1: AGE DISTRIBUTION

Age ranged from 8 to 59 years. With mean age of 29.31+/-10.75. And median age of 27years.

Table 2: SEX DISTRIBUTION

There were 109(60.9%) females and 70(39.1%) were males, with female predominance.

Table 3: ETIOLOGY

Rheumatic heart disease was the predominant contributor in this study with 173(96.6%), followed by degenerative 5(2.8%) and one case of congenital cleft mitral leading to regurgitation (6%).

Table 4: DIAGNOSIS

Mitral stenosis was the predominant lesion 84(46.9%), followed by mitral regurgitation 46(25.7%). Combined lesion accounted for 29(16.2%) and Mitral restenosis subjected to MVR was 20(11.2%).

Table 5: CO MORBID CONDITIONS

LA clot was the major associated comorbid condition which accounted for 21 (56.8%) for which Clot removal with concomitant ligation of Left Atrial Appendage done.

ASD were Five (13.5%). Three patients had undergone single vessel Coronary artery disease (8.1%). Three patients had Severe Tricuspid Regurgitation (8.1%). Two patient presented with infective endocarditis (5.4%) and another one person had a LA Myxoma (2.7%).

Table 6: PREVIOUS SURGERY

Closed Mitral Commissurotomy were done in 22 (12.3%) of patients. The prime indication now for re operation is severe mitral restenosis.

Table 7: ELECTIVE AND EMERGENCY MVR

Out of 179 patient only three(1.7%) emergency surgery undertaken. Of which two(1.12%) patients were post closed mitral commissurtomy in cardiogenic shock hence MVR done in same sitting. One patient who had LA myxoma was also taken as an emergency. Remaining 176(98.3%) of patients were taken electively.

Table 8: PREOP NYHA

Patients presented commonly with class 3 symptoms 142(79.3%), and 31 patients were in class 4 (17.3%). Two patients post closed mitral commissurotomy were in Class 5 (1.1%). Combined class 3 and 4 presentation accounted 173(96.6%).

Table 9: PRE OPERATIVE RHYTHM - NSR / AF

Majority of the patients were in NSR 109(60.9%) & AF accounted for 70(39.1%).

Table 10: PRE. OPERATIVE EJECTION FRACTION %

About 170(95%) of patients had Ejection Fraction 50 % and above. Only nine (5%) were between 30 – 49 % of Ejection Fraction.

Table 11:PROCEDURES

Isolated MVR was done in 144 (80.4%), when combined with LA Clot removal it accounted 165 (92.1%). LA clot was removed with concomitant ligation of Left Atrial Appendage in these patients. Five (2.8%) ASD Patients had, along with valve replacement ASD Pericardial patch closure .Three(1.7%) patients had undergone single vessel CABG for Coronary artery disease. Three(1.7%) patients who had Severe Tricuspid Regurgitation undergone MVR with DEVEGA Annuloplasty . One person had a LA Myxoma excision (0.6%) , combined with valve replacement. Two(1.1%) patients were post closed mitral commissurtomy in cardiogenic shock hence MVR done in same sitting.

TABLE 12 :CLASSICAL / CHORDAL PRESERVATION

Out of 179 patients 96(53.6%) had Classical MVR where all chordal attachment was excised before valve replacement. And 83(46.4%) had chordal preservation either partial or total.

TABLE 13: VALVE SIZE

In view of majority of stenotic lesion 27mm St.Jude valve was maximum used which accounts for 92(51.4%). This is followed by 25 mm valve 66(36.9 %).

TABLE 14: AF / NSR POST OPERATIVEPERIOD

The commonest post operative rhythm is NSR 110(61.5%) and AF accounted for 67(37.4%). Two patients who were in junctional rhythm and bradycardia were supported with Epicardial Pacing both of them who had LV Rupture.

TABLE 15 :POST OPERATIVE EJECTION FRACTION %

In the immediate post operative period pre discharge Echo evaluation showed 129(77.7%) were having ejection fraction 50 % and above. And 37(22.3%) of the patients were having EF% between 30-49%. This drop in EF% could be explained by majority of the Procedure were Classical in view of agglutinated, fibrotic chordae , papillary muscles & preservation in this cases were difficult.

TABLE 16: EF%-PREOP &EF%-POST OP CROSSTABULATION- CLASSICAL GROUP

This comparison was done on those patients who had undergone classical repair and alive.

Of the total 83 patients who are alive, preoperatively 80 patients had EF 50% and above and 3 patients had EF between 35-49%. Out of these 80 patients who had preop EF 50 & above , only 49 (61.3%) maintained their preop EF of 50 & above. And 31(38.3%) patients had EF dropped to 35 – 49%. And those three patients who had pre op Moderate LV Dysfunction EF (35-49) % did not show any improvement in the post op.

TABLE 17: EF-PREOP & EF% - POST OP CROSSTABULATION- CHORDAL PRESERVATION GROUP

This comparison was done on those patients who had undergone Chordal repair and alive. Of the total 83 patients who were alive, preoperatively 81 patients had EF in 50% and above and 2 patients had EF between 35-49%. Out of these 81 patients who had preop EF 50 & above, 78 (96.3%) maintained their preop EF of 50 & above. And 3(3.7%) patients had EF dropped to 35 – 49%. And those two patients who had pre op moderate LV Dysfunction EF (35-49) % showed improvement in the post op.

TABLE18 : POST OP NYHA

Majority of the patients had a definitive symptomatic improvement and 117 (65.4%) were in NYHA class 2 and 28 (15.6%) were in NYHA class 3 respectively. About 19 (10.6%) were in NYHA class 1. And one (0.6%). patient was in congestive failure in class 4.

TABLE 19 : COMPLICATIONS

Commonest complication post cardiac surgery here was myocardial failure with low cardiac output 8(4.5%), all the patient died in postoperative period. Next in the line was valve thrombosis 6(3.4%) of this ,3(1.7%) patients died. Two (1.1%) patients had thrombo emboli , both had diminished right vision and blindness. Another Two (1.1%) had prosthetic valve endocarditis. LV Rupture was seen in two (1.1%) and two (1.1%) of patient had to under go re exploration for increased inter costal drainage. One (0.6%) died of sudden cardiac arrest due to ventricular arrhythmia. Other than the wound infection , transient elevation of LFT in two patients in the immediate post bypass period settled on its own.

TABLE 20 : COMPLICATION AMONG 'NSR' PATIENTS

Majority of sinus rhythm patient had no complications 94(86.2%). Three patients had superficial wound infection(2.8%) and two patients had thromboembolism (1.8%) and another two patients had prosthetic valve endocarditis (1.8%). one patient had myocardial failure and low cardiac output syndrome(0.9%), and another one patient died

of mediastinitis(0.9%).Ventricular arrythmia occured in one patient in post operative period and died(0.9%). Bleeding events occured in three (2.8%) patients were of trivial in nature, which was managed conservatively.

TABLE 21 : COMPLICATIONS AMONG AF PATIENTS :

Majority of the patients with AF had died of myocardial failure and low cardiac output syndrome 7(10%), followed by valve thrombosis in six (8.6%)patients, two patients had LVrupture intra operatively and one patient had Left atrial appendage tear and died intraoperatively. Two patients were explored for increased intercostal drainage. One patient died intraoperatively, due to coagulation failure. Decubitus ulcer and superficial skin wound infection occured in one patient each. one patient was in congestive cardiac failure postoperatively. one patient had major bleeding episode and was admitted to the hospital . About 47(67.1%) of patients had no complications.

TABLE 22 : CAUSE OF DEATH

The leading cause of death in this study was myocardial failure with Low cardiac output syndrome 8(4.5%). This was followed by valve thrombosis 3(1.7%). Prosthetic valve endocarditis & LV Rupture caused two(1.1%) deaths each. Mediastinitis, LA Appendage tear, coagulation failure ,bleeding and Arrythmia all contributed 1(0.6%) each.Over all mortality due to all causes was 19 (10.8%). And operative mortality was 14 (7.8%).

DISCUSSION

The patients age ranged between 8-59 in this series, with a mean age of 29.31+/-10.75 and a median 0f 27 years, comparable to Choudhary etal study of rheumatic population with mean age was 22.8 +/- 11.3 years with range, 2 to 70 years. (54)

In Emery,etal study (13) the mean age was 60+/-12.8 which was on the higher side, which implies that Rheumatic etiology is a major factor in Indian scenario. Kirklin described an accelerated form of RHD in certain ethnic group with early onset of manifestation due to prior severe attack of RF (55). The majority of patients in this study were females with Mitral stenosis being the major lesion caused by Rheumatic etiology.

According to Ikonomidis etal study 60% were females affected by Rheumatic etiology and Mitral regurgitation being the major presentation .(14)

In Demirag etal study, 58.7% were females. The reason for operation was combined mitral stenosis plus regurgitation in 36.96% and etiology being acute rheumatic fever in 75.36%. (17).

The predominant comorbidity in this study was LA clot. Khalaf etal in their study concluded LA clot being predictor of inhospital mortality in MVR . Since large clot adherent to the left atrium & appendage takes longer time for removal which increases the ischemic time, cardio pulmonary bypass time at the same time predisposing to injury to the friable left atrial wall. Friable materials may embolise in the immediate post operative period.(56)

Coronary artery disease is an important long term predictor of post operative outcome. In this series out of three coronary artery disease patients operated one patient with diabetes died because of mediastinitis and sternal infection, another patient died of myocardial failure with low cardiac output in the post operative period.

According to Losanoff etal infection of median sternotomy wound leads to mortality rate between 14 and 47% secondary to underlying mediastinitis .Risk factors for sternal wound dehiscence includes diabetes mellitus, chronic obstructive airway disease, obesity, smoking, Prolonged bypass time, excessive bone wax, too much use of diathermy, all of which can inhibit wound healing, with increased transfusions , reexploration, prolonged postoperative ventilation and longer stay in the ICU leading to increased dehisence.(57).

Herlitz etal proposed mitral valve surgery in combination with CABG was independently associated with increased death and higher rehospitalization.(58)

According to Ashraf etal higher inhospital mortality for combined CABG and MVR patients depends on NYHA functional class left ventricular global wall motion score (increased scores indicating impaired function) and prolonged cross-clamp time.(59)

Emergency MVR was done in two Acute Mitral regurgitation patients post closed mitral commissurotomy , the patients were in low cardiac out put and acute

pulmonary edema. Immediately on the table conversion to MVR was done, there was no pre operative waiting period for these patients, of these two patient one died in the post operative period due to ventricular arrythymia.

Combined procedure carries higher mortality compared to isolated procedure.

Ibrahim etal in their study identified age, previous cardiac operations, diabetes mellitus, extent of coronary artery disease, preoperative NYHA class, and additional procedures as independent prognostic factors for overall survival. (18)

Previous cardiac surgery was a predictor of inhospital mortality. In our series out of total mortality of 19 patients six patients died intraoperatively or in the immediate post operative period due to redo Mitral surgery. Dense adhesions of previous surgery prolongs the intraoperative time, ischeamic time and cardiopulmonary bypass time, high risk for LV Rupture and LA Appendage damage.

Rutledge etal in their series found that patients undergoing Mitral valve replacement after closed mitral commissurotomy had a Perioperative mortality of 13%.(60)

Sampath Kumar etal in their experience with redo mitral surgery had early mortality of 5.64% with hemorrhage and low cardiac output being the major causes of death . On follow-up they found no late deaths in the valve repair group.(61)

Treasure etal enumerated the causes for LVdisruption in MVR as, heavily calcified annulus ,too large a valve implantataion, strut near A-V groove and previous cardiac surgery. (62)

Compared to preoperative functional status the post operative cardiac status improved significantly. In the Pre operative period 96.6% were in NYHA class 3 or 4, and in the post operative period 76% were in class 1 or 2.

Majority of the patient in this series presented very late with higher NYHA 3 or 4 class. Various studies have clearly pointed out the need to intervene early, before patient moves to higher NYHA class and LV deterioration..

Lee etal reported from their study that, Preoperative NYHA class III or IV symptoms and left ventricular impairment were independent risk factors for death and myocardial failure. Early mortality was lower in the valve repair and MVR-subvalvular preservation group compared to Classical MVR group. Since myocardial failure is the main cause of death in mitral valve surgery , subvalvular preservation reduced the severity of myocardial failure rather than preventing it . Not only will Conservative surgery optimizes the outcome but the early surgery before severely limiting symptoms or LV impairment gives better outcome.(63).

In this study , patients who had undergone chordae preservation majority maintained their post operative EF% in the normal range.

Chronic AF is not a benign rhythm in the long term. In this study all the post operative valve thrombosis patients were in AF preoperatively Except for one, all the others were in fast ventricular response at time of admission with thrombosed valve.

Valve thrombosis patients presented from two to six months after post MVR. At the time of admission all six were in sub therapeutic level of anticoagulation. One patient presented clinically with stroke , one with TIA which was evaluated and diagnosed. Remaining patients presented in NYHA class 4 symptoms. One patient died of stroke , other two patients died of cardiac failure.

Deviri etal in their study with valve thrombosis patients found that the time interval between valve replacement and obstruction ranged from 6 weeks to 13 years (median 4 years). Of 63% patients in whom coagulation profile was available at the time of obstruction, 70% were receiving inadequate anticoagulant. About 63% of the patient were in New York Heart Association functional class IV.(37)

Lafci etal from their study concluded that subtherapeutic anticoagulation level was the major etiologic factor involved in the pathogenesis of valve Thrombosis . Thrombosis occurred in the mitral position in (78%), compared to aortic position in (22%) in their series. The mean duration from valve replacement to valve thrombosis was 48.3 +/- 15.4 months. The majority of patients presented with poor functional status (56% in NYHA class IV) and poor anticoagulation (INR < or = 2 in 72% of cases) (35).

Of the two thromboembolic episode and fatal loss of vision one patient presented in the early post operative period, probable cause would be particulate debris or air emboli. Other patient at time of admission had low INR level.

One patient presented in this series with anticoagulant related hemorrhage as gastrointestinal bleed which required admission and blood and factors transfusion. Other presented with minor bleeding event required anticoagulant modification strategies.

Remadi etal in their series had problem maintaining target INR, hence they advocated for a lighter anticoagulant strategy for patients in sinus rhythm with self monitoring of INR like in blood glucose monitoring in home. (15).

In this series two patients had prosthetic valve endocarditis , microbial etiology in one being affected by candida and other being affected by acineto bacter baumannii. Both patients had blood culture positive for organism at different occasions. Acinetobacter baumannii infected patient presented after two months with sternal infection.

According to AliIlgin etal Acinetobacter is a highly resistant microorganism, commonly isolated in intensive and post-operative care units. It may constitute one of the several causes of early prosthetic valve endocarditis. A diffuse, red maculopapular rash may be encountered in patients with Acinetobacter endocarditis.(64)

Of the overall mortality of 19 patients , 14 (73.68 %)of them were in AF and 5(26%) in NSR.

SUMMARY

Between June 2005 and December 2006 , 179 St Jude mechanical mitral valves were implanted. Age ranged from 8 to 59years, mean age 29.31+/-10.75 with median of 27 years. Majority being female 109(60.9%) with Rheumatic heart disease 173(96.6%) being the commonest etiology with Mitral stenosis 84(46.9%) as the major lesion . overall mortality was 19(10.8%) and in hospital mortality was 14(7.8%) and about 75% of the patients were still in regular follow up. LA clot 21(56.8%) being the major co -morbid condition followed by ASD 5(13.5%) and TR & CAD 3(8.1%) constituted each. One patient had LA Myxoma 1(2.7%); Mitral restenosis 20(11.2%). NYHA class preoperatively 3 and 4 combined173(96.6%). Most patients were in NSR109(60.9%) and AF (39.1%) with majority having LVEF fifty and above 170(95%). Isolated MVR 144(80.4%) being the commonest procedure , CABG & Devega annuloplasty being 3 (1.7%) each. Classical procedure MVR 96(53.6%) and chordal preservation in 83(46.4%)only. Post operatively out of 80 Pre. op normal EF patients only49(61.3%) had normal EF in the classical group. And in the chordal preservation group out of 81 pre op. normal EF patients 78(96.3%) had normal EF postoperatively . Most of valve replacement patients had definitive symptomatic improvement in NYHA class, majority were in NYHA 1 and 2 combined 136(76%).

Majority of the patient were in Sinus Rhythm 110(61.5%) , AF 67(37.4%). There was no structural valve failure, no paravalvular leak or valve related hemolysis.

There were 6 (3.4%) valve thrombosis; prosthetic valve endocarditis 2(1.1%); anticoagulant related hemorrhage 4(2.2%); thromboembolism 2(1.1%); myocardial failure 8(4.5%); LV rupture 2(1.1%).

High NYHA Class , AF, associated CAD, Redo mitral surgery were significant risk factors for inhospital mortality and the presence of AF was a significant risk factor for increased mortality and morbidity on follow up.

CONCLUSION

Mitral valve replacement cannot normalize the life expectancy. Valve replacement has its own limitations. Population coming for mitral valve replacement are under privileged , and they lack financial backup to manage the post valve replacement sequelae. Valve replacement is not the end of the disease but a new beginning of a chronic disease lying dormant , ready to blow off at any time, given a chance. The best way to address this issue is to stress the importance of mass education, school health education, strict implementation of secondary prophylaxis and an early clinical trial for vaccines for rheumatic fever. In failed cases earlier intervention should be done and not to wait until the heart fails, or AF sets in. Timely intervention when the valve is suitable for conservative procedures and to promote valve sparing surgery as much as possible, there by avoiding the prosthetic valve. In the established cases conservative surgery in the form of chordal preservation should be done with antiarrythymic surgery whenever possible.

A diligent search should be made to find a valve substitute that lacks the problems , presently available prosthesis have.

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